PERIODONTAL EMERGENCIES IN GENERAL PRACTICE

ABSTRACT

Diagnosing and managing periodontal emergencies is a common part of general dental practice. This article summarises the presentation, aetiology and management of the key periodontal emergencies including: gingival abscess, periodontal abscess, peri-coronitis/peri-coronal abscess, perio-endo lesion/abscess, necrotising gingivitis and periodontitis, acute herpetic gingivostomatitis, acute physical/chemical/thermal injury and subgingival root fracture.

KEY WORDS

Periodontal emergency; Acute periodontal conditions

INTRODUCTION

A periodontal emergency arises when an acute condition involving the periodontium causes pain, forcing the patient to seek urgent care. The general dentist is usually the first port of call and so should be confident in accurately assessing and managing the emergency. Timely management is key in preventing further damage to the periodontium as well as improving the patient's physical and psychological well-being.

This article provides an overview of the key periodontal emergencies and discusses their presentation, aetiology and management.

GINGIVAL ABSCESS

A gingival abscess is 'a localised purulent infection that involves the marginal gingiva or interdental papillae'[1] *(Figure 1)*.

Presentation

The clinical features will include a rapidly expanding localised area of swelling that may be shiny and smooth or pointed. Suppuration may also be present. The lesion is usually painful for the patient and tender to palpation.

Aetiology

Gingival abscesses are often associated with subgingivally impacted foreign objects and tend to occur in a previously healthy gingiva.[2] A variety of foreign bodies have been described in the literature including pieces of nails in subjects with a nail-biting habit.[3] The term “oral hygiene abscesses’’ has been proposed for abscesses caused by the impaction of foreign bodies that are oral hygiene aids.[4]

Management

The abscess should be incised, drained and irrigated with saline to relieve the acute symptoms. Mitigation of the aetiology is an important consideration and mechanical debridement may help with this. Short-term use of chlorhexidine mouthwash or warm saline rinses is often recommended, especially if the area is too tender to brush. A follow-up appointment should be arranged to check for resolution.

PERIODONTAL ABSCESS

A periodontal abscess is defined as ‘a localised accumulation of pus within the gingival wall of a periodontal pocket resulting in the destruction of the collagen fibre attachment and the loss of nearby alveolar bone’[5]. In a study performed in general practice in the UK, 6–7% of patients treated in one month suffered from a periodontal abscess, which made it the third most prevalent infection that demanded emergency treatment after dento-alveolar abscesses and pericoronitis.[6]

Presentation

The most prominent sign is the presence of an ovoid elevation in the gingiva along the lateral part of the root. However, abscesses deep in the periodontium may be less apparent presenting as a diffuse swelling or simply a red area. The area may be tender on touch. The abscess is usually associated with a deep periodontal pocket with bleeding and tenderness on probing. Suppuration may also occur through a fistula or through the periodontal pocket opening and may be spontaneous or following finger pressure. Increased tooth mobility and tenderness on percussion are common.[7] The patient may also report the tooth feeling “high” in the occlusion. As periodontal abscesses are usually associated with existing periodontal pockets, some degree of bone loss around the tooth in question is likely to be seen radiographically. When performing sensibility testing, the tooth should respond positively.

Aetiology

A periodontal abscess may represent disease exacerbation of existing periodontitis in the presence of complex pocket morphology, furcation involvement or a vertical defect, in which marginal closure of the pocket may lead to spread of infection into the surrounding periodontal tissues. Changes in the composition of the subgingival microbiota, with an increase in bacterial virulence, or a decrease in the host defence, may also result in a diminished capacity to drain the increased suppuration.[8] Common examples of when this may occur include:

- Following debridement - calculus fragments may become dislodged and pushed into the periodontal tissues.[9]
- After surgical therapy - associated with the presence of foreign bodies such as membranes or sutures.[10]
- Acute exacerbation of periodontitis.[11]
- Systemic antimicrobial intake without subgingival debridement in severe periodontitis (related to an overgrowth of opportunistic bacteria).[12]

Management

Treatment involves two distinct phases: control of the acute condition to arrest tissue destruction and control the symptoms followed by management of a pre-existing and/or residual lesion, especially in patients with periodontitis. If the tooth has advanced bone loss or is heavily restored, the most sensible treatment option may be extraction. If the tooth can be saved, drainage needs to be established either through the pocket or with an external incision and the periodontal pocket should be thoroughly debrided. Occlusal adjustment may help to provide immediate relief. Systemic antibiotics are only required if there are clear signs of systemic involvement or spreading infection. As most periodontal abscesses occur in a pre-existing periodontal pocket, periodontal therapy should be evaluated after resolution of the acute phase. In cases where the patient has not been treated previously, the appropriate periodontal treatment should be provided. If the patient is already within the active phase of therapy, the periodontal therapy should be completed once the acute lesion has been treated. In patients receiving supportive periodontal therapy, careful evaluation of the recurrence of the abscess should be made, as well as assessment of the tissue damage and its implications on the tooth's long-term prognosis.[8]

PERI-CORONITIS/ PERI-CORONAL ABSCESS

Pericoronitis is 'inflammation of the soft tissues surrounding the crown of a partially erupted tooth'[13]. A peri-coronal abscess is the 'localised accumulation of pus within the overlying

gingival flap surrounding the crown of an incompletely erupted tooth'[5].

Presentation

Red, swollen, possibly suppurative lesion that is painful to touch. Swelling of the cheek at the angle of mandible, trismus and a radiating pain to ear are common. The patient may also experience systemic complications such as lymphadenopathy, fever and general malaise.

Aetiology

The partially erupted and impacted mandibular third molar is the site most frequently involved. The overlying operculum is an excellent harbour for the accumulation of debris and bacteria. In addition, insult to the operculum is often produced by trauma from an opposing tooth.

Management

Following anaesthesia, the operculum should be irrigated to remove any debris. In some cases, it may be necessary to excise the operculum and possibly also perform some occlusal adjustment on the opposing tooth to eliminate any sources of trauma. Antibiotics are only indicated if there are systemic signs or spreading infection. Once the acute phase of the infection has subsided, a decision needs to be made on whether the tooth requires extraction. If the tooth is retained, optimal oral hygiene measures are necessary to help prevent further acute pericoronitis episodes. The evidence suggests that 'the first episode of pericoronitis, unless particularly severe, should not be considered as an indication for surgery. Second or subsequent episodes should be considered appropriate indication for surgery.'[14] Extraction might also be indicated if the tooth is associated with extensive caries or root resorption.

PERIO-ENDO LESION/ABSCESS

Combined periodontal/endodontic lesions are localised, circumscribed areas of infection originating in the periodontal and/or pulpal tissues. They are essentially as a result of a communication between the periodontal pocket and the pulp.

Presentation

The abscess will be associated with a deep pocket surrounding a non-vital tooth. The pocket may circumscribe a large part of the tooth or be a localised narrow deep lesion. A smooth, shiny swelling of the gingiva or mucosa will be present. The swelling may have purulent exudate or fistula and will be tender to palpation. The tooth may be tender to percussion and

mobile. A typical radiographic appearance is shown in *Figure 2*.

Aetiology

The infection may arise primarily from pulpal inflammatory disease expressed through the periodontal ligament or the alveolar bone to the oral cavity. Or it may be initiated from a periodontal pocket communicating to the pulp apically or through accessory canals.[1] Vertical root fracture of a tooth (which may not be heavily restored or root canal treated) may also present in a similar manner. Rapid loss of the periodontal attachment and peri-radicular tissues may ensue.

Management

Considerations include establishing drainage by debriding the pocket and/or by incising the abscess. Other treatments may include endodontic therapy, irrigation of the pocket, limited occlusal adjustment and/or the administration of antimicrobials if there are signs of spreading infection and management of patient comfort. If the lesion is primary endodontic, it often responds well to root canal therapy only. If the lesion is primary periodontal or truly combined there is often a very poor prognosis and this is usually a good indication for extraction in single rooted teeth. In multi-rooted teeth, root resection may a possible consideration following endodontic therapy. It is also necessary to ensure that there has not been a vertical fracture, since this may often render a tooth unsavable.

Table 1 summarises the key features of the gingival, periodontal, peri-coronal and perio-endo abscess.

NECROTISING PERIODONTAL DISEASES

Necrotising periodontal diseases are considered the most severe inflammatory lesions associated with the oral biofilm.[15] These include necrotising gingivitis (NG) and necrotising periodontitis (NP).[16] NG describes a scenario where only the gingival tissues are affected *(Figure 3)*. In NP, the necrosis progresses into the periodontal ligament and alveolar bone, leading to attachment loss *(Figure 4)*. It has been suggested that these conditions may be different stages of the same disease.[17]

Presentation

The mandibular anterior teeth are most commonly affected. NG will be associated with necrosis and ulcers in the free gingiva. These lesions start at the interdental papilla and typically have a ‘punched out’ appearance. Marginal erythema may be present and necrotic

lesions can progress to the marginal gingiva. A pseudo-membrane may form over the necrotic area. When this 'membrane' is removed, the underlying connective tissue becomes exposed and bleeds. The severity of pain experienced by the patient is dependent on the severity and extension of the lesions. The bouts of pain usually increase with eating and oral hygiene practices. Other less common symptoms include halitosis, fever and malaise. NP will have the features described for NG but in addition the necrosis will affect the periodontal ligament and alveolar bone, leading to attachment loss. As the disease progresses, an interproximal crater will divide the buccal and lingual/palatal portion of the papilla. If these craters are deep, the interdental crestal bone may become denuded. When interproximal necrotic areas spread laterally and merge, this results in the creation of an extensive zone of destruction. In severe cases, bone sequestration may occur. In patients with NG, there may be associated risk factors such as high stress levels, heavy smoking and poor nutrition. Both NG and NP may be associated with untreated HIV/AIDS and other diseases (and occasionally drug regimes), which compromise the immune system.

Aetiology

Necrotising periodontal diseases are caused by infectious but commensal organisms. However, predisposing factors including a compromised host immune response are key in facilitating bacterial pathogenicity. Knowledge of the pathogenesis of this condition is limited but the spirochetes and fusiform bacteria described in the necrotic lesions have been shown to have the capacity to invade the epithelium[18] and connective tissue[19], as well as release endotoxins, which may cause periodontal tissue destruction through modification of the host response.

Management

Superficial debridement to remove soft and mineralised deposits should be carefully performed. Ultrasonic rather than hand instruments are recommended to ensure minimum pressure over the ulcerated soft tissue. The debridement may be performed daily, getting deeper as the patient's tolerance improves, lasting for as long as the acute phase lasts (usually 2-4 days). Mechanical oral hygiene measures such as brushing should be limited to avoid pain and ensure healing. The patient should be advised to use chemical plaque control agents, such as chlorhexidine mouthwash (0.2% twice daily) instead. Other agents such as 3% hydrogen peroxide diluted in 1:1 warm water and other oxygen-releasing agents provide an additional antibacterial effect against anaerobes through the release of oxygen.[20] If response to debridement is poor or if there are systemic signs, the use of systemic antimicrobials should be considered. Metronidazole (400 mg TDS for 5 days) is usually the first choice due to its action against strict anaerobes.[21] As signs and symptoms improve,

strict oral hygiene measures should be enforced and debridement should be completed where necessary. Once the acute phase has been controlled, treatment of any pre-existing periodontal disease and control of systemic risk factors should be addressed. These patients require close monitoring and support. Longer-term management may involve attempts to try to disguise soft tissue asymmetry, which may be present following resolution of lesions.

ACUTE HERPETIC GINGIVOSTOMATITIS

Herpetic gingivostomatitis is the most common viral infection of the oral mucosa *(Figure 5)*.

Presentation

This condition is characterised by small ulcers with elevated margins that may be dispersed throughout the mouth on both attached and unattached mucosal surfaces. The patient may experience generalised pain in the gingiva and/or oral mucosa. Systemic signs such as lymphadenopathy, fever and malaise may also be present. Lesions normally last for 7–10 days and heal without scarring. This condition is more frequently observed in children 2 to 5 years of age.

Aetiology

Herpetic gingivostomatitis is caused by the herpes simplex virus (HSV-1). It is the initial presentation during the primary herpes simplex infection and is of greater severity than herpes labialis, which is often the subsequent presentation.

Management

As the condition is self-limiting, no treatment is usually indicated beyond hydration and management of symptoms. However, if the condition presents in immune-compromised patients, antivirals agents such as acyclovir, may be used topically and/or systemically to control the infection. Palliative therapy to relieve pain must be initiated to allow the patient to eat and drink.

The key differences between necrotising gingivitis and acute herpetic gingivostomatitis are summarised in Table 2.

ACUTE PHYSICAL/CHEMICAL/THERMAL INJURY

This group of acute periodontal lesions are not aetiologically associated with the oral biofilm. However, it is important to be familiar with these as they are not uncommon and accurate

diagnosis will ensure appropriate therapeutic management.

Presentation

- Physical injuries may appear as erosions or ulcers and can be associated with gingival recession. Less frequently they can present as hyperkeratosis, vesicles or bullae. If the physical trauma is limited but continuous over time, the lesion may be hyperkeratotic. If the trauma is more aggressive, superficial laceration may occur. The lesion may be either asymptomatic or cause an intense localised pain in the area of the lesion.
- Thermal injury is usually painful for the patient and the appearance of the gingival tissues may be erythematous, desquamated and occasionally associated with vesicles, erosions or ulcers.
- Chemical lesions may appear after direct contact of the agent over the mucosa, leading to maculae, vesicles, erosions or ulcers, depending on the causal agent and the duration of contact *(Figure 6)*.[8]

Aetiology

- Physical injuries are most commonly caused by inappropriate oral hygiene habits, traumatic injuries or parafunction.[22-24] For example, the use of an abrasive dentifrice and an overzealous horizontal brushing technique might result in an ulcer or erosion in the gingiva. The incorrect use of dental floss or other interdental devices may also result in gingival ulceration, inflammation and recession *(Figure 7)*. Physical injuries can be self-induced and examples of traumatic agents include fingers, nails or items such as pencils *(Figure 8)*. Self-induced injuries are more frequent in children or teenagers, sometimes with psychological conditions.[25,26] Traumatic injuries might also be related to fractured teeth, orthodontic appliances or oral piercings.[27,28]
- Common causes of chemical trauma are oral bleaching agents, either due to inappropriate use by the patient or poorly fitting trays. Other agents include etchants and less commonly used dental products.[29-31]
- Thermal injury is commonly related to burns caused by very hot food or drinks.

Management

Therapeutic intervention will depend upon the diagnosis and cause. As well as clinical examination, an accurate patient history will be important in determining the source of the trauma. Treatment will include elimination of the initiating factor if required and symptomatic management of the pain. Lesions usually heal without further intervention but on some

occasions additional treatment may be required. It is important to distinguish this group from lesions related to mucocutaneous diseases where there may not be a clear initiating factor. Hence it may be necessary to withhold mechanical oral hygiene procedures supplemented by the use of chemical plaque control to help establish aetiology.

SUBGINGIVAL ROOT FRACTURES

A fracture of a tooth extending from the supragingival oral environment in an apical direction subgingivally can give rise to acute pain and periodontal infection.

Presentation

Magnification and good illumination can help to visualise fracture lines. A “tooth sleuth” or similar device may also aid diagnosis by applying occlusal loads to individual cusps. Even if a fracture line cannot be visualised, fractured teeth are characteristically associated with a localised deep pocket depth and possibly an abscess. The tooth / fractured cusp may be extremely tender to percussion, or pain may only be felt on release of biting load. Fractures may be vertical along the root axis, or at an angle with varying degrees of root involvement.

Aetiology

The patient may/may not be aware of a specific traumatic event during chewing. Heavily restored teeth without cuspal coverage and bruxists are key risk factors. This is also not uncommon in patients with a reduced periodontium due to an unfavourable crown:root ratio.

Management

Management depends on the vitality of the tooth as well as the location and extent of the fracture. It may be necessary to remove existing restorations followed by careful assessment of the tooth to confirm diagnosis. Treatment options usually involve endodontic treatment followed by a full coverage restoration if the tooth is restorable. A periodontal flap can be useful in visualising the fracture. Crown lengthening can help to expose the most apical extent of the fracture. In some cases, the tooth will be deemed untreatable and will require extraction *(Figure 9)*.

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TABLES & FIGURES ATTACHED SEPARATELY